Common Types of Supraventricular Tachycardia: Diagnosis and Management

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The most common types of supraventricular tachycardia are caused by a reentry phenomenon producing accelerated heart rates. Symptoms may include palpitations (including possible pulsations in the neck), chest pain, fatigue, lightheadedness or dizziness, and dyspnea. It is unusual for supraventricular tachycardia to be caused by structurally abnormal hearts. Diagnosis is often delayed because of the misdiagnosis of anxiety or panic disorder. Patient history is important in uncovering the diagnosis, whereas the physical examination may or may not be helpful. A Holter monitor or an event recorder is usually needed to capture the arrhythmia and confirm a diagnosis. Treatment consists of short-term or as-needed pharmacotherapy using calcium channel or beta blockers when vagal maneuvers fail to halt or slow the rhythm. In those who require long-term pharmacotherapy, atrioventricular nodal blocking agents or class Ic or III antiarrhythmics can be used; however, these agents should generally be managed by a cardiologist. Catheter ablation is an option in patients with persistent or recurrent supraventricular tachycardia who are unable to tolerate long-term pharmacologic treatment. If Wolff-Parkinson-White syndrome is present, expedient referral to a cardiologist is warranted because ablation is a potentially curative option. (*Am Fam Physician.* 2010;82(8):942-952. Copyright © 2010 American Academy of Family Physicians.)

▶ **Patient information:** A handout on supraventricular tachycardia, written by the authors of this article, is provided on page 956.

Supraventricular tachycardia (SVT) is tachycardia having an electropathologic substrate arising above the bundle of His and causing heart rates exceeding 100 beats per minute. Accelerated rhythms can be frightening to the patient if recurrent or persistent, and can cause significant morbidity. This article focuses on the most common types of paroxysmal SVT: atrioventricular nodal reentrant tachycardia (AVNRT), atrioventricular reciprocating tachycardia (AVRT), and atrial tachycardia (AT). Although atrial fibrillation and flutter are classified as types of SVT, they will not be discussed in this article and are reviewed elsewhere.[1,2] *Table 1* lists the common types of SVT and usual characteristics.[3-6] *Figure 1* depicts AVNRT, AVRT, AT, and normal sinus rhythm.

Epidemiology

SVTs (excluding atrial fibrillation or flutter and multifocal AT) have an estimated incidence of 35 per 100,000 person-years, with a prevalence of 2.29 per 1,000 persons.[7] Although AVNRT is the most common SVT in adults (approximately 50 to 60 percent),[4] AVRT is most common in children (accounts for approximately 30 percent of all SVTs).[4,5]

Pathophysiology

AVNRT and AVRT are electrical aberrancies that occur mainly as a result of reentry. Less commonly, increased automaticity or triggered activity can be the mechanism and usually results in a narrow complex tachycardia. AT can result from one of the three mechanisms *(Table 1)*.[3-6] AVNRT and AVRT are atrioventricular nodal-dependent arrhythmias, whereas AT is an atrioventricular nodal-independent arrhythmia.

AVNRT

The most common type of SVT is AVNRT. Most patients with AVNRT do not have structural heart disease; the group most often affected is young, healthy women.[8] However, some patients do have underlying heart disease, such as pericarditis, previous myocardial infarction, or mitral valve prolapse.[9] The coexistence of slow and fast pathways in atrioventricular nodal tissue is the basis of aberrant substrate for reentrant tachyarrhythmias.[10]

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SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	*Evidence rating*	*References*
Intravenous adenosine (Adenocard) or verapamil is a safe and effective treatment choice for terminating SVT, but verapamil is more effective for suppression of this rhythm over time.	B	2, 14
Vagal maneuvers are an effective first-line treatment option for SVT in younger patients who are hemodynamically stable; they can also be diagnostic for nodal-dependent SVT.	C	2, 21
Brugada criteria are sensitive and specific in helping distinguish between SVT with aberrancy and ventricular tachycardia.	C	33
Adenosine may be used as a diagnostic or therapeutic agent in patients with undifferentiated wide complex tachycardia.	C	35
Radiofrequency ablation is a safe, effective, and cost-effective method for suppressing SVT, and it improves patient quality of life compared with medical treatment of SVT.	B	2, 41, 42

SVT = supraventricular tachycardia.

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to http://www.aafp.org/afpsort.xml.

Table 1. Common Types of Supraventricular Tachycardia and Usual Characteristics

Type	*Epidemiology*	*Mechanism*	*Possible electrocardiography changes*
AVNRT	Most common SVT (approximately 50 to 60%)[4] Occurs more often in younger women	Reentry caused by nodal pathways or tracts (two types): atypical (fast/slow) represents 10% and typical (slow/fast) represents 90% of all AVNRT	Rate: 118 to 264 bpm Rhythm: regular, narrow QRS complex (< 120 msec); regular, wide QRS complex (≥ 120 msec); may not see any P-wave activity in either type (atypical or typical) Atypical AVNRT: RP interval > PR interval; P waves negative in leads III and aVF Typical AVNRT: RP interval < PR interval; pseudo R wave in lead V_1 with tachycardia, not with normal sinus rhythm; pseudo S wave in leads I, II, and aVF
AVRT	Second most common SVT (approximately 30%)[4,5] Orthodromic most common type (81 to 87%) Occurs more often in younger women and children May be comorbid with Wolff-Parkinson-White syndrome	Reentry caused by accessory pathways (two types): orthodromic (antegrade conduction through atrioventricular node) and antidromic (retrograde conduction through atrioventricular node)	Rate: 124 to 256 bpm Rhythm: regular, narrow QRS complex common (orthodromic); regular, wide QRS complex uncommon (orthodromic or antidromic) if bundle branch block or aberrancy present Orthodromic AVRT: RP interval < PR interval *or* RP interval > PR interval with a slowly conducting accessory pathway; retrograde P waves (leads I, II, III, aVF, V_1); delta wave seen with normal sinus rhythm, not with tachycardia Antidromic AVRT: short RP interval (< 100 msec); regular, wide QRS complex (≥ 120 msec); delta waves seen with normal sinus rhythm and tachycardia; concealed accessory pathways do not show delta waves
AT	Third most common SVT (approximately 10%)[6] Two types: AT and multifocal AT AT has two forms: focal and macroreentrant Multifocal AT occurs more often in middle age or in persons with heart failure or chronic obstructive pulmonary disease	Reentry (micro), automaticity, or triggered activity: focal AT (reentry, automaticity, or triggered activity); multifocal AT (automaticity activity)	Rate: 100 to 250 bpm (atrial); ventricular varies Rhythm: regular, narrow QRS complex usually; irregular (ectopic foci) may have wide QRS complex if aberrancy present Focal AT: long RP interval most common; P-wave shape/polarity variable Multifocal AT: three different P-wave morphologies exist unrelated to each other; RR interval irregularly

AT = atrial tachycardia; AVNRT = atrioventricular nodal reentrant tachycardia; AVRT = atrioventricular reciprocating tachycardia; bpm = beats per minute; SVT = supraventricular tachycardia.

Information from references 3 through 6.

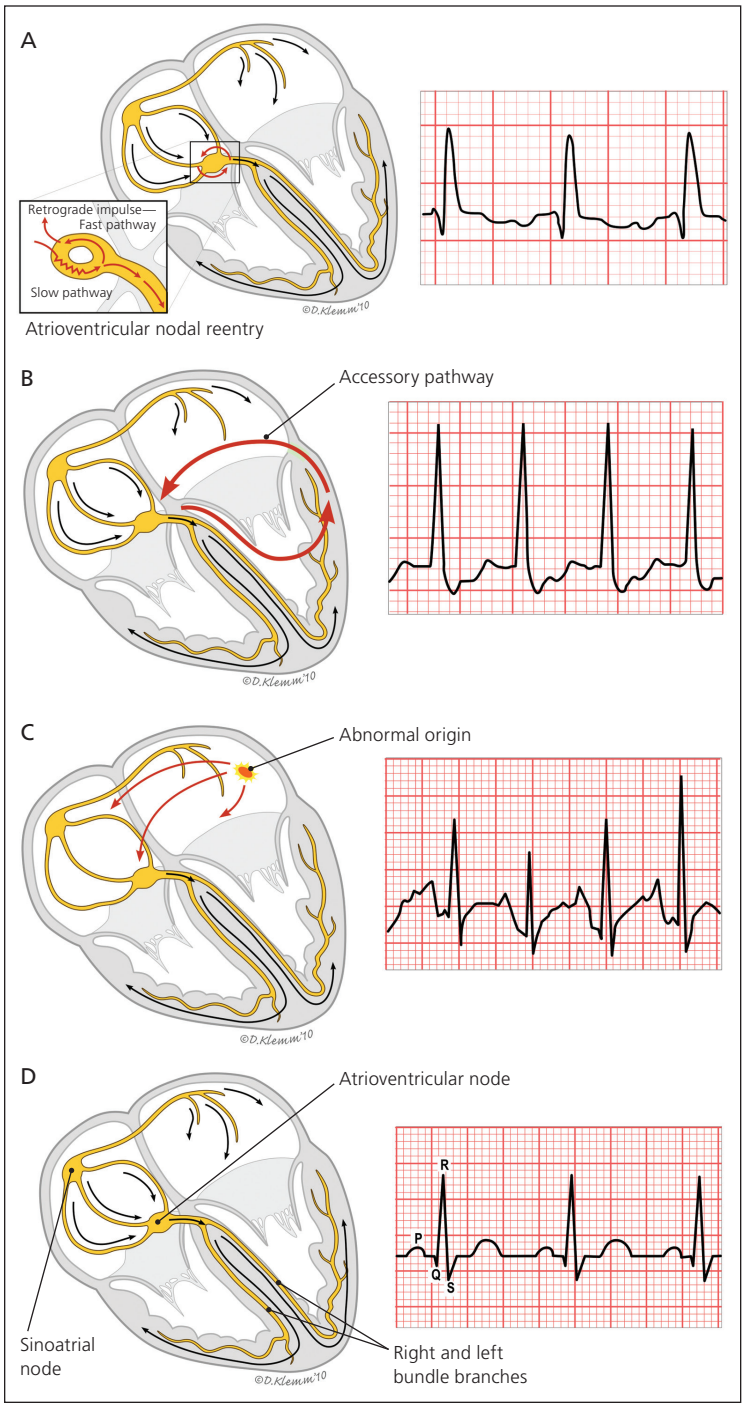

Figure 1. *(A)* In typical atrioventricular nodal reentrant tachycardia (antegrade conduction down the slow atrioventricular nodal pathway and retrograde conduction up the fast pathway), the retrograde P wave may not be seen or may be visible early after the QRS complex. When visible, it often appears as a pseudo R wave in lead V_1. *(B)* In atrioventricular reciprocating tachycardia, there is typically a short RP interval, with the timing and morphology of the P wave dependent on the site and conduction velocity of the accessory pathway. *(C)* Atrial tachycardia typically produces variable RP and PR intervals because atrioventricular conduction depends on atrioventricular nodal properties and the tachycardia rate. In atrial tachycardia, the morphology and axis of the P wave are influenced by atrial site of origin and tachycardia mechanism. Short- and long-term therapies are discussed in the text. *(D)* Normal sinus rhythm.

AVRT

The second most common type of SVT is AVRT. Patients with this arrhythmia typically present at a younger age than those with AVNRT. This SVT is caused by accessory pathways (or bypass tracts) that serve as aberrant conduits for impulses that pass from the sinoatrial node and travel in an antegrade or retrograde fashion through such tracts, establishing a reentry circuit.[11] AVRT, occasionally comorbid with Wolff-Parkinson-White syndrome, is a diagnosis not to be missed because this rhythm may spontaneously develop into atrial fibrillation.[12] Key electrocardiography (ECG) findings, such as a delta wave, are not always apparent because of the accessory pathway being concealed; therefore, special diagnostic testing may be needed.[13]

AT

The third most common type of SVT is AT (approximately 10 percent); it originates from a single atrial focus.[6] This SVT, if focal, usually has a definitive localized origin, such as adjacent to the crista terminalis in the right atrium or the ostia of the pulmonary veins in the left atrium.[14,15] Another form, multifocal AT, often occurs in patients with heart failure or chronic obstructive pulmonary disease.[16]

Patient Evaluation

HISTORY AND PHYSICAL EXAMINATION

Symptoms of SVT depend on a number of factors, including patient age, presence of comorbid heart and lung disease, and duration of SVT episodes. *Table 2* lists symptoms associated with SVT. Patients may also be asymptomatic or minimally symptomatic, potentially delaying diagnosis.

The history may reveal the likely etiology underlying the SVT *(Table 3)*. Sinus tachycardia must be considered in the differential diagnosis. Episodic SVT may be misdiagnosed as anxiety or panic disorder,[17] especially in patients with a psychiatric history, prolonging definitive diagnosis and treatment. Prolonged and persistent elevated heart rates produced by some types of SVT have been known to cause a type of cardiomyopathy; therefore, a high index of suspicion for the diagnosis is important.[18]

Table 2. Symptoms Usually Associated with Supraventricular Tachycardia

Common
Chest discomfort or pressure; dyspnea; fatigue; lightheadedness or dizziness; palpitations (including possible pulsations in the neck)
Uncommon
Chest pain (more severe than discomfort); diaphoresis; nausea; presyncope or syncope
Rare
Sudden death (may occur with Wolff-Parkinson-White syndrome)

The physical examination may or may not be helpful in determining a possible etiology for a patient's symptoms. Younger patients who are otherwise healthy usually have a normal examination, with tachycardia (if present on examination) being the only physical finding. *Table 4* lists items to include in a focused examination and diagnostic workup.

ECG

A 12-lead ECG should be performed in patients who are hemodynamically stable, with special attention to rhythm and rate, atrioventricular conduction (PR interval), RP interval, hypertrophy, pathologic Q waves, prolongation of the QT interval, and any evidence of preexcitation. *Figures 2 through 5* are example ECGs for the types of SVT discussed. Most types of SVT have narrow QRS complexes. Wide complex tachyarrhythmias can also occur and can be secondary to SVT associated with bundle branch block, an accessory pathway, or ventricular tachycardia. In patients with a history of (or suspected) coronary artery disease or myocardial infarction, wide complex tachyarrhythmias must be considered to be of ventricular origin until proven otherwise and treated as such (see the treatment section). *Table 1* describes ECG findings for common types of SVT.[3-6]

Further clinical investigations and their possible significance to SVT should be pursued *(Table 4)*. Patients should be expediently referred to a cardiologist or electrophysiologist if they have experienced syncope or severe dyspnea, or if preexcitation is present on resting 12-lead ECG. *Table 5* lists other situations in which patients should be referred to a cardiologist or electrophysiologist.

Treatment

The primary treatment goal for any SVT is its cessation, especially in patients who are at risk hemodynamically and cannot tolerate prolonged tachyarrhythmias. SVT may be rare and fleeting in some patients, whereas in others, it is more frequent and may cause serious symptoms such as presyncope or syncope. Treatment of SVT can be divided into short-term or urgent management and long-term management.

SHORT-TERM OR URGENT MANAGEMENT

Short-term or urgent management of SVT can be separated into pharmacologic and nonpharmacologic strategies. Nonpharmacologic management typically uses maneuvers that increase vagal tone to decrease heart rate. Pharmacologic management typically includes intravenous adenosine (Adenocard) or verapamil, which are safe

Table 3. History in Patients with Possible Supraventricular Tachycardia

Inquiry	*Possible implication*
At what age did the symptoms begin (time of onset)?	Symptoms since early childhood suggest supraventricular tachycardia
Did symptoms begin when patient was sedentary or active?	Coronary ischemia with activity may lead to ventricular problems
How did the symptoms begin (gradually or suddenly)?	Sinus tachycardia starts and stops gradually
What were the symptoms (e.g., syncope, presyncope, lightheadedness with rapid heart rate, dizziness, shortness of breath, palpitations)?	Any combination of these symptoms suggests supraventricular tachycardia, especially in patients with Wolff-Parkinson-White syndrome
How long did the symptoms last?	Supraventricular tachycardia starts and stops quickly (within seconds)
What were the potential triggers (e.g., caffeine, reduced sleep, increased stress)?	Increased sympathetic discharge may induce sinus tachycardia
Is there a cardiac history?	Symptoms or arrhythmias after myocardial infarction or ischemia suggest ventricular origin
Is there a family history of cardiac disease or sudden death?	Ischemia or any sudden death suggest supraventricular tachycardia
Has the patient had any cardiac procedures?	History of ischemic heart disease is consistent with ventricular issues

Table 4. Physical Examination and Diagnostic Workup in Patients with Possible SVT

Evaluation	*System or test*	*Possible finding*	*Significance*
Focused physical examination	Cardiovascular	Murmur(s)	Valvular heart disease causing heart failure or tachycardia
		Friction rub	Pericarditis resulting in tachycardia
		Third heart sound	Heart failure causing tachycardia
		Cannon waves	Possible atrioventricular nodal reentrant tachycardia or ventricular tachycardia
	Respiratory	Crackle	Heart failure resulting in tachycardia
	Endocrine	Enlarged or tender thyroid gland	Hyperthyroidism or thyroiditis resulting in tachycardia
In-office testing	Vitals	Hemodynamic instability or febrile illness	Incite tachyarrhythmia
	Orthostatic blood pressure	Autonomic or dehydration issues	Induce tachyarrhythmia
	Electrocardiography	Preexcitation	Wolf-Parkinson-White syndrome
		Wide versus narrow QRS complex	Type of SVT versus ventricular tachycardia
		Q waves	Ischemia leading to ventricular tachycardia
		Other findings	Type of SVT (see Table 1)
Blood work	Complete blood count	Anemia or infection	All possibly induce or incite tachyarrhythmia
	Thyroid-stimulating hormone	Suppression or hyperthyroidism	
	Basic metabolic panel	Electrolyte disturbance	
	B-type natriuretic peptide	Congestive heart failure	
	Cardiac enzymes	Myocardial infarction or ischemia	
Diagnostics	Chest radiography	Cardiomegaly	Congestive heart failure or cardiomyopathy
	Holter monitor or event recorder	Capture aberrant rhythm, frequency, duration	Type of tachyarrhythmia
	Graded exercise test	Preexcitation or aberrant rhythm	Type of tachyarrhythmia
	Echocardiography	Structural or valvular disease	Possible surgical intervention

SVT = supraventricular tachycardia.

and effective treatment choices for terminating SVT, but verapamil is more effective for suppression of this rhythm over time.[2,14] *Figure 6* is an algorithm for the short-term management of SVT.[19] Patients who are hemodynamically unstable need to be resuscitated with electrocardioversion to avoid further deterioration of cardiovascular status.

If the patient is hemodynamically stable, the QRS complex can provide information in decision making. A narrow QRS complex (less than 120 msec) usually indicates SVT, and the Valsalva maneuver is the most widely used and feasible treatment option in an alert patient. Although the use of this technique has been accepted in hospitalized settings, it has not been studied in the prehospital setting to determine its effectiveness.[20] Vagal maneuvers are an effective first-line treatment option for SVT in younger patients who are hemodynamically stable; they can also be diagnostic for nodal-dependent SVT.[2,21] Carotid massage can be used as a diagnostic and therapeutic tool; however, it should not be used in persons who may have atherosclerotic plaque that could be dislodged as a result of such a technique (i.e., history of carotid artery disease or carotid bruit).[21]

If these methods fail to terminate the SVT, or if the SVT soon returns, pharmacologic therapy is used. *Table 6* shows recommended agents for short-term management of SVT.[22] Which agent is selected after use of vagal maneuvers and adenosine depends on patient factors, such as contraindications (any comorbid conditions or allergies), hemodynamic stability, or presence of a wide QRS complex.

Adenosine is an atrioventricular nodal blocking agent with a very short half-life (nine to 12 seconds). It is highly effective for the termination of nodal-dependent SVT and is the first-line drug for acute conversion of narrow complex SVT.[23] Adenosine has the advantage of temporarily slowing the rate enough to determine the underlying focus of the rhythm (i.e., ventricular or supraventricular). Adenosine should not be used in

Figure 2. Electrocardiogram of a narrow complex tachycardia with a 1:1 atrioventricular association in a 16-year-old girl with tachypalpitations. The differential diagnosis includes atrial tachycardia, atrioventricular nodal reentrant tachycardia, and orthodromic atrioventricular reciprocating tachycardia. Rhythm was terminated with 6 mg of intravenous adenosine (Adenocard).

Figure 3. Postconversion electrocardiogram demonstrating the typical features of ventricular preexcitation with short PR interval and prominent delta wave. This finding supports orthodromic atrioventricular entry as the likely mechanism of supraventricular tachycardia. Diagnostic electrophysiology confirmed the mechanism.

Figure 4. Electrocardiogram of a narrow complex tachycardia with a 1:1 atrioventricular association. This example represents atrioventricular reciprocating tachycardia, which has a high cure rate with catheter ablation therapy.

Figure 5. Electrocardiogram of a narrow complex tachycardia with atrioventricular association and right bundle branch block aberration. This example represents atrioventricular nodal reentrant tachycardia, which is also depicted in Figure 1A.

Table 5. Situations in Which Referral to a Cardiologist or Electrophysiologist Is Warranted

Referral is warranted if:
Medications are not controlling symptoms
Patient cannot tolerate medications or no longer wishes to use them
Patient has worsening symptoms or is becoming hemodynamically unstable
Patient is in a high-risk occupation (e.g., pilot, truck driver, heavy equipment operator) or participates in high-risk recreational activities (e.g., rock climbing, sky or scuba diving)
Physician is uncomfortable with or uncertain about management or initial diagnosis
Preexcitation is present on electrocardiography or if atrioventricular reciprocating tachycardia is suspected
Supraventricular tachycardia is accompanied by syncope
Wide QRS complex is present on electrocardiography

persons with Wolff-Parkinson-White syndrome and atrial fibrillation because this rhythm can degenerate into ventricular fibrillation.[24] There is poor evidence that medical therapy reduces sudden death in patients with Wolff-Parkinson-White syndrome; therefore, expedient referral for ablation is recommended in those patients who are symptomatic.[2,25] Because AT is an atrioventricular nodal-independent SVT, atrioventricular nodal blocking agents, such as verapamil or adenosine, are mostly ineffective in its termination.[26]

Verapamil is a calcium channel blocker that may be used in patients with SVT that recurs after adenosine therapy. Verapamil, a negative inotrope, can result in relative bradycardia and vasodilation; care must be used if patients have a significant decrease in cardiac output.[27] Neither digoxin nor a calcium channel blocker should be used in patients with Wolff-Parkinson-White syndrome or wide complex tachycardia, because these agents may enhance conduction down the accessory pathway, predisposing the patient to ventricular fibrillation.[28] If the SVT persists, addition of a beta blocker will often result in its termination.[29]

In a review of eight trials involving 577 patients, there was no difference in the effectiveness of adenosine versus verapamil in successfully treating SVT. The overall termination rate was about 90 percent for both agents.[30] If SVT is not terminated and the patient is hemodynamically stable, flecainide (Tambocor) or propafenone (Rythmol) may be used to terminate the SVT.[31,32] These agents are almost always administered by a cardiologist.

Compared with narrow complex tachycardia, wide complex tachycardia presents infrequently, but does occur under certain conditions. Wide complex tachycardia is often difficult to distinguish from ventricular

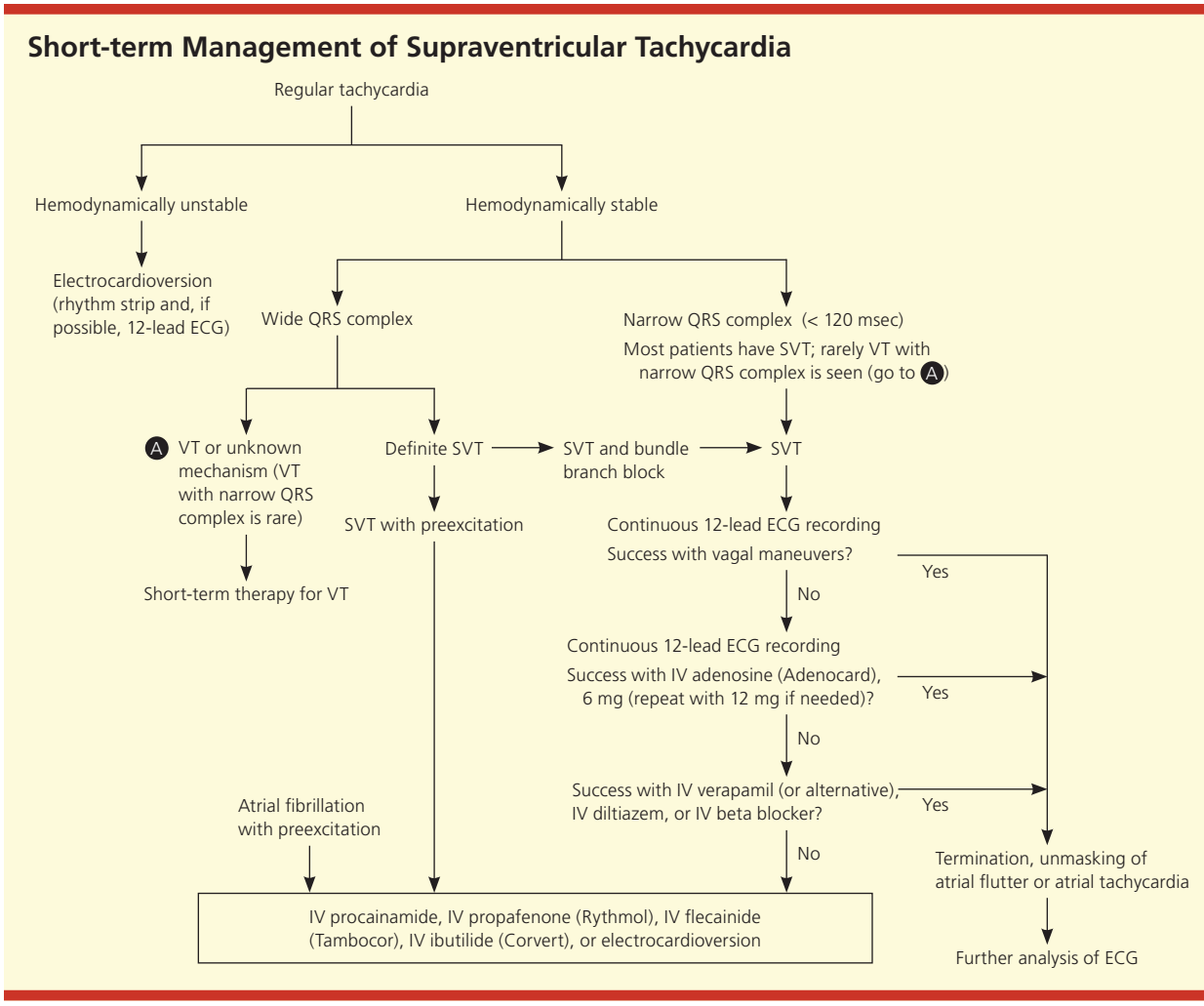
Short-term Management of Supraventricular Tachycardia
Regular tachycardia
Hemodynamically unstable
Hemodynamically stable
Electrocardioversion (rhythm strip and, if possible, 12-lead ECG)
Wide QRS complex
Narrow QRS complex (< 120 msec)
Most patients have SVT; rarely VT with narrow QRS complex is seen (go to A)
A
VT or unknown mechanism (VT with narrow QRS complex is rare)
Definite SVT
SVT and bundle branch block
SVT
SVT with preexcitation
Continuous 12-lead ECG recording
Success with vagal maneuvers?
Yes
No
Short-term therapy for VT
Continuous 12-lead ECG recording
Success with IV adenosine (Adenocard), 6 mg (repeat with 12 mg if needed)?
Yes
No
Success with IV verapamil (or alternative), IV diltiazem, or IV beta blocker?
Yes
No
Atrial fibrillation with preexcitation
Termination, unmasking of atrial flutter or atrial tachycardia
IV procainamide, IV propafenone (Rythmol), IV flecainide (Tambocor), IV ibutilide (Corvert), or electrocardioversion
Further analysis of ECG

Figure 6. Algorithm of the short-term management of supraventricular tachycardia (SVT). (ECG = electrocardiography; IV = intravenous; VT = ventricular tachycardia.)

Adapted from Delacrétaz E. Clinical practice. Supraventricular tachycardia. N Engl J Med. *2006;354(10):1044.*

tachycardia, and all types should be treated as ventricular tachycardia when SVT cannot be discerned, particularly in patients who are hemodynamically unstable. If the patient is hemodynamically stable, use of the well-known Brugada criteria *(Table 7)* can help distinguish between SVT with aberrancy and ventricular tachycardia, with a reported sensitivity as high as 98.7 percent and specificity as high as 96.5 percent.[33] More recently, a newer simplified algorithm based on the Brugada criteria has been proposed. It may be more accurate in determining true ventricular tachycardia, with a reported overall test accuracy of 90.3 percent compared with 84.8 percent when all four Brugada criteria are used.[34]

A recent retrospective study showed that intravenous adenosine used in 197 patients with undifferentiated wide complex tachycardia was safe and effective for diagnostic and therapeutic purposes. No adverse effects occurred, and the likelihood of making a correct diagnosis of SVT or ventricular tachycardia increased.[35]

LONG-TERM MANAGEMENT

The long-term management of SVT is based on the SVT type; frequency and intensity of the episodes; overall impact on the quality of life of the patient; and risks of the therapy chosen.[19] Discussion of these issues with the patient will help determine the optimal treatment strategy. The primary options include catheter ablation (radiofrequency versus cryotherapy) or pharmacologic treatment *(Table 6)*.[22] *Figure 7* is an algorithm for the long-term management of SVT.[19]

Patients with infrequent SVT episodes may only need pharmacotherapy on an intermittent basis, or what has been described as the "pill-in-the-pocket" approach.[36] Those experiencing SVT not more than a few times per year, but with episodes lasting one hour or longer, may be treated using this approach. This is typically done with verapamil (40 to 160 mg) in patients without preexcitation or a beta blocker in patients without chronic obstructive pulmonary disease or asthma.

Table 6. Short- and Long-term Management Options for Supraventricular Tachycardia

Agent name	*Class*	*Indication*	*Dosage*	*Comments*
Short-term				
Adenosine (Adenocard)	V: endogenous purine nucleotide	Terminates SVT	6 mg rapid intravenous push, repeat with 12 mg if needed	Can be diagnostic and therapeutic Extremely short half-life Adverse effects include chest pain and dyspnea during administration Contraindicated in patients with Wolff-Parkinson-White syndrome
Diltiazem	IV: calcium channel blocker	Decreases rate	0.25 mg per kg intravenous bolus	Adverse effects include dizziness, heart failure exacerbation Avoid in patients with Wolff-Parkinson-White syndrome or wide complex tachycardia
Esmolol (Brevibloc)	II: beta blocker	Decreases rate	500 mcg per kg intravenous loading dose	Can be proarrhythmic; has short half-life Avoid in patients with renal disease; use with care in patients with asthma
Verapamil	IV: calcium channel blocker	Decreases rate	5 mg intravenously, up to 15 mg	Avoid in patients with congestive heart failure, Wolff-Parkinson-White syndrome, wide complex tachycardia, or atrioventricular block (second or third degree)
Long-term				
Amiodarone (Cordarone)	III: potassium channel blocker*	Prevents SVT	200 to 400 mg orally once daily	Can result in optic neuritis, thyroid dysfunction, pulmonary fibrosis
Disopyramide (Norpace)	Ia: sodium channel blocker*†	Prevents SVT	200 to 400 mg orally twice daily	Adverse effects include urinary retention
Metoprolol	II: beta blocker	Decreases rate	25 to 100 mg orally twice daily	Atrioventricular node suppression possible
Procainamide	Ia: sodium channel blocker	Prevents SVT (long-term use)	250 to 500 mg orally every six hours	Adverse effects include lupus, hypotension, His-Purkinje block
Quinidine	Ia: sodium channel blocker*†	Prevents SVT (long-term use)	324 to 648 mg orally every eight to 12 hours	Avoid in patients with atrial fibrillation because of increased mortality Closely monitor QTc interval when initiating therapy
Verapamil	IV: calcium channel blocker	Prevents SVT	80 to 240 mg orally three times daily	Adverse effects include constipation, dizziness Avoid in patients with Wolff-Parkinson-White syndrome or wide complex tachycardia

SVT = supraventricular tachycardia.

**—May prolong QT interval.*

†—*Widens QRS complex.*

Information from reference 22.

Table 7. Original Brugada Criteria for Assessing Wide Complex Tachyarrhythmias

Electrocardiographic change	*Criterion present*	*Criterion not present*
1. RS complex absent from all precordial leads	VT present; stop and treat	Proceed to 2
2. RS complex is present, and the longest precordial RS interval is > 100 msec in one or more precordial leads	VT present; stop and treat	Proceed to 3
3. Atrioventricular dissociation is present	VT present; stop and treat	Proceed to 4
4. Morphologic criteria for VT* present in precordial leads V_1 to V_2 *and* V_6	VT present; stop and treat	Supraventricular tachycardia with aberrant conduction is diagnosis made by exclusion

VT = ventricular tachycardia.

**—For complete morphologic criteria, see Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex.* Circulation. *1991;83(5):1649-1659.*

Information from reference 33.

Long-term Management of SVT

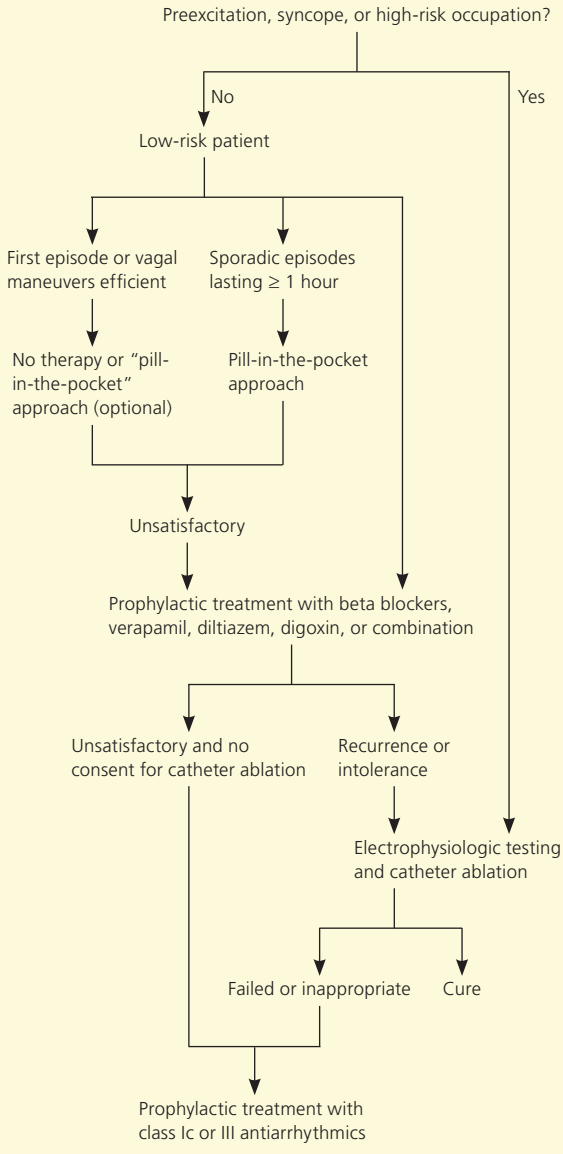
Preexcitation, syncope, or high-risk occupation?
No
Yes
Low-risk patient
First episode or vagal maneuvers efficient
Sporadic episodes lasting ≥ 1 hour
No therapy or "pill-in-the-pocket" approach (optional)
Pill-in-the-pocket approach
Unsatisfactory
Prophylactic treatment with beta blockers, verapamil, diltiazem, digoxin, or combination
Unsatisfactory and no consent for catheter ablation
Recurrence or intolerance
Electrophysiologic testing and catheter ablation
Failed or inappropriate
Cure
Prophylactic treatment with class Ic or III antiarrhythmics

Figure 7. Algorithm of the long-term management of supraventricular tachycardia (SVT).

Adapted from Delacrétaz E. Clinical practice. Supraventricular tachycardia. N Engl J Med. *2006;354(10):1046.*

Table 8. Vaughan-Williams Classification of Antiarrhythmics

Class	*Description*
Ia	Reduce maximum velocity (rate of rise of action potential upstroke [phase 0])
	Prolong action potential duration
	Kinetics of onset and offset in blocking the sodium channel are of intermediate rapidity (less than five seconds)
	Examples include quinidine, procainamide, disopyramide (Norpace)
Ib	Do not reduce maximum velocity
	Shorten action potential duration
	Kinetics of onset and offset in blocking the sodium channel are rapid (less than 500 msec)
	Examples include mexiletine, phenytoin (Dilantin), lidocaine (Xylocaine)
Ic	Can reduce maximum velocity
	Primarily slow conduction
	Prolong refractoriness minimally
	Kinetics of onset and offset in blocking the sodium channel are slow (10 to 20 seconds)
	Examples include flecainide (Tambocor), propafenone (Rythmol)
II	Block beta-adrenergic receptors
	Examples include propranolol (Inderal), timolol, metoprolol
III	Predominantly block potassium channels (e.g., inward rectifier potassium channels)
	Prolong repolarization
	Examples include sotalol (Betapace), amiodarone (Cordarone)
IV	Predominantly block the slow calcium channel (e.g., L-type calcium channel)
	Examples include verapamil, diltiazem, nifedipine (Procardia), felodipine (blocks T-type calcium channel)
V	Have various mechanisms of action
	Examples include adenosine (Adenocard), digoxin, magnesium sulfate

Information from reference 22.

Agents used for long-term pharmacotherapy are similar to those used to terminate the SVT during short-term management. Atrioventricular nodal blocking agents (e.g., verapamil, diltiazem, beta blockers, digoxin) in AVNRT and AVRT with retrograde conduction are only about 30 to 60 percent effective.[37] This relative lack of effectiveness can necessitate use of two such agents or the addition of class Ic or III antiarrhythmics. Larger trials comparing outcomes between these drug classes are not yet available *(Table 8[22])*. Generally, these agents should be managed by a cardiologist.

Ablative therapy of SVT is based on the observation that most arrhythmias arise from a focal origin critically dependent on conduction through a defined anatomic structure. If those critical regions are destroyed,

the arrhythmia no longer occurs spontaneously or with provocation. Because of shorter procedure duration, lessened fluoroscopic exposure, and increased knowledge in this area of cardiology, catheter ablation is becoming the first-line treatment option for all patients with SVT, not just those with symptomatic arrhythmias refractory to suppressive drug therapy or those who prefer a drug-free lifestyle.

Clinical series of radiofrequency catheter ablation of accessory pathways have been published with excellent overall results.[38] Experienced electrophysiology laboratories routinely achieve success rates of 95 percent in the ablation of accessory pathways, with recurrence rates of less than 5 percent.[39] With improved knowledge of atrioventricular nodal anatomy and the advent of cryotherapy ablation, the current rate of symptomatic heart block is 0.5 to 1 percent.[40]

Because of its curative results and low percentages of severe adverse effects, and because the field is evolving so rapidly, there are few studies directly comparing catheter ablation with drug therapy in patients with SVT (with the exception of atrial fibrillation). However, observational studies have reported that patients undergoing radiofrequency ablation for SVT have better overall quality-of-life outcomes and lower costs attributed to therapy compared with medical treatment.[41,42]

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REFERENCES

1. Fuster V, Rydén LE, Cannom DS, et al. ACC/AHA/ESC 2006 guidelines for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (writing committee to revise the 2001 guidelines for the management of patients with atrial fibrillation): developed in collaboration with the European Heart Rhythm Association and Heart Rhythm Society [published correction appears in *Circulation.* 2007;116(6):e138]. *Circulation.* 2006;114(7):e257-e354.
2. Blomström-Lundqvist C, Scheinman MM, Aliot EM, et al. ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol.* 2003;42(8):1493-1531.
3. Kumar UN, Rao RK, Scheinman MM. The 12-lead electrocardiogram in supraventricular tachycardia. *Cardiol Clin.* 2006;24(3):427-437, ix.
4. Porter MJ, Morton JB, Denman R, et al. Influence of age and gender on the mechanism of supraventricular tachycardia. *Heart Rhythm.* 2004; 1(4):393-396.
5. Ko JK, Deal BJ, Strasburger JF, Benson DW Jr. Supraventricular tachycardia mechanisms and their age distribution in pediatric patients. *Am J Cardiol.* 1992;69(12):1028-1032.
6. Wellens HJ, Brugada P. Mechanisms of supraventricular tachycardia. *Am J Cardiol.* 1988;62(6):10D-15D.
7. Orejarena LA, Vidaillet H Jr, DeStefano F, et al. Paroxysmal supraventricular tachycardia in the general population. *J Am Coll Cardiol.* 1998; 31(1):150-157.
8. Rodriguez LM, de Chillou C, Schläpfer J, et al. Age at onset and gender of patients with different types of supraventricular tachycardias. *Am J Cardiol.* 1992;70(13):1213-1215.
9. Akhtar M, Jazayeri MR, Sra J, Blanck Z, Deshpande S, Dhala A. Atrioventricular nodal reentry. Clinical, electrophysiological, and therapeutic considerations. *Circulation.* 1993;88(1):282-295.
10. Ganz LI, Friedman PL. Supraventricular tachycardia. *N Engl J Med.* 1995; 332(3):162-173.
11. Chauhan VS, Krahn AD, Klein GJ, Skanes AC, Yee R. Supraventricular tachycardia. *Med Clin North Am.* 2001;85(2):193-223, ix.
12. Mark DG, Brady WJ, Pines JM. Preexcitation syndromes: diagnostic considerations in the ED. *Am J Emerg Med.* 2009;27(7):878-888.
13. Katoh T, Ohara T, Kim EM, Hayakawa H. Non-invasive diagnosis of concealed Wolff-Parkinson-White syndrome by detection of concealed anterograde pre-excitation. *Jpn Circ J.* 2001;65(5):367-370.
14. Roberts-Thomson KC, Kistler PM, Kalman JM. Focal atrial tachycardia II: management. *Pacing Clin Electrophysiol.* 2006;29(7):769-778.
15. Kistler PM, Roberts-Thomson KC, Haqqani HM, et al. P-wave morphology in focal atrial tachycardia: development of an algorithm to predict the anatomic site of origin. *J Am Coll Cardiol.* 2006;48(5):1010-1017.
16. Kastor JA. Multifocal atrial tachycardia. *N Engl J Med.* 1990;322(24): 1713-1717.
17. Lessmeier TJ, Gamperling D, Johnson-Liddon V, et al. Unrecognized paroxysmal supraventricular tachycardia. Potential for misdiagnosis as panic disorder. *Arch Intern Med.* 1997;157(5):537-543.
18. Fenelon G, Wijns W, Andries E, Brugada P. Tachycardiomyopathy: mechanisms and clinical implications. *Pacing Clin Electrophysiol.* 1996;19(1): 95-106.
19. Delacrétaz E. Clinical practice. Supraventricular tachycardia. *N Engl J Med.* 2006;354(10):1039-1051.
20. Smith G, Morgans A, Boyle M. Use of the Valsalva manoeuvre in the prehospital setting: a review of the literature. *Emerg Med J.* 2009;26(1):8-10.
21. Adlington H, Cumberbatch G. Carotid sinus massage: is it a safe way to terminate supraventricular tachycardia? *Emerg Med J.* 2009;26(6):459.
22. Libby P, ed. *Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine.* 8th ed. Philadelphia, Pa.: W.B. Saunders; 2007.
23. DiMarco JP, Miles W, Akhtar M, et al. Adenosine for paroxysmal supraventricular tachycardia: dose ranging and comparison with verapamil. Assessment in placebo-controlled, multicenter trials. The adenosine for PSVT study group [published correction appears in *Ann Intern Med.* 1990;113(12):996]. *Ann Intern Med.* 1990;113(2):104-110.

24. Belardinelli L, Linden J, Berne RM. The cardiac effects of adenosine. *Prog Cardiovasc Dis*. 1989;32(1):73-97.
25. Marine JE. Catheter ablation therapy for supraventricular arrhythmias. *JAMA*. 2007;298(23):2768-2778.
26. Glatter KA, Cheng J, Dorostkar P, et al. Electrophysiologic effects of adenosine in patients with supraventricular tachycardia. *Circulation*. 1999;99(8):1034-1040.
27. Fox DJ, Tischenko A, Krahn AD, et al. Supraventricular tachycardia: diagnosis and management. *Mayo Clin Proc*. 2008;83(12):1400-1411.
28. Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ. Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *N Engl J Med*. 1979;301(20):1080-1085.
29. Anderson S, Blanski L, Byrd RC, et al. Comparison of the efficacy and safety of esmolol, a short-acting beta blocker, with placebo in the treatment of supraventricular tachyarrhythmias. The Esmolol vs Placebo Multicenter Study Group. *Am Heart J*. 1986;111(1):42-48.
30. Holdgate A, Foo A. Adenosine versus intravenous calcium channel antagonists for the treatment of supraventricular tachycardia in adults. *Cochrane Database Syst Rev*. 2006;(4):CD005154.
31. Pritchett EL, Wilkinson WE. Mortality in patients treated with flecainide and encainide for supraventricular arrhythmias. *Am J Cardiol*. 1991; 67(11):976-980.
32. Reimold SC, Maisel WH, Antman EM. Propafenone for the treatment of supraventricular tachycardia and atrial fibrillation: a meta-analysis. *Am J Cardiol*. 1998;82(8A):66N-71N.
33. Brugada *P*, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. *Circulation*. 1991;83(5):1649-1659.
34. Vereckei A, Duray G, Szénási G, Altemose GT, Miller JM. Application of a new algorithm in the differential diagnosis of wide QRS complex tachycardia. *Eur Heart J*. 2007;28(5):589-600.
35. Marill KA, Wolfram S, Desouza IS, et al. Adenosine for wide-complex tachycardia: efficacy and safety. *Crit Care Med*. 2009;37(9):2512-2518.
36. Alboni P, Tomasi C, Menozzi C, et al. Efficacy and safety of out-of-hospital self-administered single-dose oral drug treatment in the management of infrequent, well-tolerated paroxysmal supraventricular tachycardia. *J Am Coll Cardiol*. 2001;37(2):548-553.
37. Winniford MD, Fulton KL, Hillis LD. Long-term therapy of paroxysmal supraventricular tachycardia: a randomized, double-blind comparison of digoxin, propranolol and verapamil. *Am J Cardiol*. 1984;54(8):1138-1139.
38. Borggrefe M, Budde T, Podczeck A, Breithardt G. High frequency alternating current ablation of an accessory pathway in humans. *J Am Coll Cardiol*. 1987;10(3):576-582.
39. Kay GN, Epstein AE, Dailey SM, Plumb VJ. Role of radiofrequency ablation in the management of supraventricular arrhythmias: experience in 760 consecutive patients. *J Cardiovasc Electrophysiol*. 1993;4(4):371-389.
40. Mitrani RD, Klein LS, Hackett FK, Zipes DP, Miles WM. Radiofrequency ablation for atrioventricular node reentrant tachycardia: comparison between fast (anterior) and slow (posterior) pathway ablation. *J Am Coll Cardiol*. 1993;21(2):432-441.
41. Goldberg AS, Bathina MN, Mickelsen S, Nawman R, West G, Kusumoto FM. Long-term outcomes on quality-of-life and health care costs in patients with supraventricular tachycardia (radiofrequency catheter ablation versus medical therapy). *Am J Cardiol*. 2002;89(9):1120-1123.
42. Cheng CH, Sanders GD, Hlatky MA, et al. Cost-effectiveness of radiofrequency ablation for supraventricular tachycardia [pubished correction appears in *Ann Intern Med*. 2001;135(10):933]. *Ann Intern Med*. 2000;133(11):864-876.